> ***is no criminal responsibility***; but in the second, responsibility never ceases.

Id.

This available concept of settled insanity would have resulted in no crimin responsibility. It also refuted the jury instructions that left the impression that M Crutsinger's intoxication was voluntary, and not to be considered.[17]

Moreover, under TEX. CODE CRIM. PROC. 38.36, Mr. Crutsinger could h introduced his evidence of "settled insanity" as "condition-of-the-mind" evidence in the guilt/innocence phase to negate the *mens rea* element of the charged offense. TEX. CODE CRIM. PROC. 38.36 provides in pertinent part:

> (a) In all prosecutions for murder, ... the defendant shall be permitted to offer testimony as to ... ***All relevant facts and circumstances going to show the condition of the mind of the accused at the time of the offense.***

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[17] The jury instructions recite:

> You are instructed that voluntary intoxication is not a defense to the commission of a crime.

(CR 7:1270).

(iii) punishment – less morally culpable

Evidence of "settled insanity" and likely brain damage had mitigating significance in the punishment phase of the trial and could have refuted the prosecutor's contention that Mr. Crutsinger was "evil." (RR 33:5). More importantly, it would have proved the non-volitional nature of Mr. Crutsinger's aggression by the showing the correlation between brain injury and antisocial behavior. This evidence would have allowed a juror to conclude that Mr. Crutsinger was less morally "culpable than defendants who have no such excuse," but who acted "deliberately" as that term is commonly understood." *Penry v. Lynaugh*, 492 U.S. 302, 322-323 (1989), *reversed in part*, *Atkins v. Virginia*, 536 U.S. 304 (2002) (holding that executing mentally retarded criminal violates the Eighth Amendment as cruel and unusual punishment).

2. Trial counsel's performance was prejudicial

As all of the aforementioned reflects, trial counsel's deficient performance resulted in prejudice.

Pre-trial Phase: The absence of evidence of Mr. Crutsinger's mental impairments pre-trial, resulted in the mistaken conclusion by the state courts' that

because the confession was voluntary, the confession, as well as all the evidence flowing from it, was admissible.

Guilt/Innocence Phase: The absence of evidence of Mr. Crutsinger's mental impairments in the guilt/innocence phase, resulted in the imposition of criminal responsibility and a directed verdict of guilt for the prosecution. This, despite the law of settled insanity,[18] and the admissibility of this evidence to negate the *mens rea* element of capital murder.[19]

Had this evidence been admitted, the "settled insanity" of Mr. Crutsinger, which arose from his habitual drunkenness since the age of 17 years, would have refuted the conclusions of law, such as CL #15 that:

> "given the gruesome circumstances of the murders, the overwhelming evidence connecting [Mr. Crutsinger] to these murders, it is not reasonable to believe that the jury would have reached a different result had Mr. Ray and Mr. Moore done more investigation, more communication, or more preparation in this case."

See also CL #15-17.

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[18] Where there is "settled insanity" due to long standing alcohol abuse, there is no criminal responsibility. " *Evers v. State*, 31 Tex. Crim.. 318, 330; 20 S.W. 744, 748 (Tex. Crim. App. 1892). *See also Thomas v. State*, 147 Tex. Crim. 45, 50; 177 S.W.2d 777, 779 (Tex. Crim. App. 1944); *Duke v. State*, 61 Tex. Crim. 441, 447; 134 S.W. 705, 708 (Tex. Crim. App. 1911).

[19] TEX. CODE CRIM. PROC. 38.36(a).

Punishment Phase: Finally, there was an abundance of mitigating evidence of Mr. Crutsinger's mental impairments that are correlated to non-volitional antisocial behaviors. But because of trial counsel's deficient performance, the jurors were left with only one explanation in the punishment phase – the argument given by the prosecutor that Mr. Crutsinger was "evil." (RR 33:5).

3. The state court determinations are contrary to, and an unreasonable application of, *Strickland*

The state court determinations that Mr. Crutsinger "was not denied effective assistance of counsel by Mr. Ray and Mr. Moore's representation," is contrary to, and an unreasonable application of, *Strickland* and its progeny. Trial counsel's ineffectiveness lay not in their failure to present evidence of Mr. Crutsinger's mental impairments in all phases of the trial, but rather in their failure to conduct a timely and adequate investigation sufficient to support the reasonableness of the "strategic" decisions they made in the defense of Mr. Crutsinger. *Wiggins v. Smith*, __U.S. __, 123 S.Ct. 2527, 2535 (2003) ("strategic choices made after less than complete investigation are reasonable precisely to the extent that reasonable professional judgments support the limitations on investigation. In other words, ***counsel has a duty***

to make reasonable investigations or to make a reasonable decision that makes particular investigations unnecessary").

Accordingly, the state court rulings are both contrary to and an unreasonable application of *Strickland* and its progeny. There is a reasonable probability that the undiscovered "evidence, taken as a whole, 'might well have influenced the jury's appraisal' of [Mr. Crutsinger's] culpability," *Wiggins v. Smith,* 539 U.S. at 538 (quoting *Williams v. Taylor,* 529 U.S., at 398), and the likelihood of a different result if the evidence had gone in is "sufficient to undermine confidence in the outcome" actually reached at sentencing, *Strickland,* 466 U.S., at 694." *Rompilla v. Beard*, 545 U.S. 374, 393 (2005). For all the aforementioned reasons, habeas relief must be granted.

GROUND THREE (HERRERA – ACTUAL INNOCENCE)

Mr. Crutsinger is actually innocent of the offense of capital murder because he lacked the necessary *mens rea* for the commission of capital murder. Accordingly, his conviction and concomitant sentence of death violate fundamental federal constitutional law principles and habeas relief must be granted. *Herrera v. Collins*, 506 U.S. 390, 417 (1993).

A. Standard of Review

In *Herrera v. Collins*, 506 U.S. 390 (1993), the United States Supreme "Court assumed without deciding that 'in a capital case a truly persuasive demonstration of 'actual innocence' made after trial would render the execution of a defendant unconstitutional, and warrant federal habeas relief if there were no state avenue open to process such a claim.'" *House v. Bell*, __ U.S. __, 126 S.Ct. 2064, 2087-88 (2006).

The Supreme Court characterized the "threshold showing for such an assumed right [as] ... necessarily ... extraordinarily high." *Herrera*, 506 U.S. at 417. However, because the *Herrera* Court left open the question whether "federal courts may entertain convincing claims of actual innocence," *Herrera*, 506 U.S. at 427, the exact parameters of the burden of proof by a defendant have yet to be defined. *House*, 126

S.Ct. at 2087 ("whatever burden a hypothetical freestanding innocence claim would require, this petitioner has not satisfied it.").

B. Mr. Crutsinger is actually innocent of capital murder

Mr. Crutsinger is actually innocent of the offense of capital murder because he lacked the necessary *mens rea* for the commission of capital murder.

1. *Mens rea* is an essential element of capital murder

The State of Texas indicted Mr. Crutsinger, alleging that:

> on or about the 6th day of April 2003, [Mr. Crutsinger] did then and there intentionally cause the death of an individual, Patricia Syren by stabbing or cutting her with a knife and did then and there intentionally cause the death of an individual, Pearl Magouirk by stabbing or cutting her with a knife and both murders were committed during the same criminal transaction

(CR 1:03).

The Charge of The Court instructed:

> ***A person commits the offense of murder if he intentionally causes the death of an individual.***
>
> ***A person commits the offense of capital murder if he commits murder*** as defined above and the person murders more than one person during the same criminal transaction.
>
> ....

> A person acts ***intentionally,*** or with intent, with respect to a result of his conduct ***when it is his conscious objective or desire to cause the result.***
>
> ....
>
> Now bearing mind the foregoing instructions, if you believe from the evidence beyond a reasonable doubt that on or about the 6th day of April, 2003, in Tarrant County, Texas, the defendant, Billy Jack Crutsinger, did then and there intentionally cause the death of an individual, Pearl Magouirk, by stabbing or cutting her with a knife and both murders were committed during the same criminal transaction, then you will find the defendant guilty of capital murder as charged in the indictment.

(CR 7:1279, 1280).

At the conclusion of the guilt/innocence phase, the jury found Mr. Crutsinger guilty of capital murder. (CR 7:1284).

2. Because of his "settled insanity," Mr. Crutsinger lacked the necessary *mens rea* for capital murder; however, there was confusion in the law at the time of trial as to whether this evidence was admissible in guilt/innocence to negate*mens rea*

There is no dispute that Mr. Crutsinger had a long-standing addiction to alcohol. He started drinking at seventeen (17) years of age, and at age forty-eight (48), approximately 30 years later, was undoubtedly addicted to alcohol. Exhibit C: Report of Dr. Goodness, dated September 10, 2003, pp.1, 6.

Further, it is very likely that his alcohol addiction resulted in brain damage, that was present on the date and at the time of the charged offenses. Exhibit C: Report of

Dr. Goodness, dated September 10, 2003, pp. 6, 12. ("When an individual has abused alcohol to the degree that that their liver is affected, chances are that their brain will also have incurred damage."). As a result, this evidence calls into question whether Mr. Crutsinger's acts of killing were "intentional" – as defined by TEXAS PENAL CODE §6.03(a) because of his "settled insanity."

In *Evers*, the Texas Court of Criminal Appeals has ruled:

> ***The first [the breaking down of the person's system by long continued or habitual drunkenness] is from drinking as a remote result,*** the second from drinking as a directresult. ***The first [***settled insanity***] is an involuntary result,*** from which all shrink alike; the second is voluntarily sought after. ***In the first*** [settled insanity***], there is no criminal responsibility;*** but in the second, responsibility never ceases.

Evers v. State, 31 Tex. Crim.. 318, 330; 20 S.W. 744, 748 (Tex. Crim. App. 1892).

Although TEX. CODE CRIM. PROC. 38.36(a) was in effect as of the date of Mr. Crutsinger's trial, arguably there was confusion in Texas law as to whether the law permitted the defense to introduce relevant mental impairment evidence in guilt/innocence to negate *mens rea*.[20]

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[20] *See* for example:

1983 *De La Garza v. State*, 650 S.W. 2d 870, 876 (Tex. App. – San Antonio 1983, no. pet.) (in attempted capital murder case, court held: "The ***defense of diminished capacity*** has not been recognized by the Texas Legislature nor by the courts.") (emphasis supplied);

1989 *Miller v. State*, 770 S.W.2d 865, 867 (Tex. App. – Austin 1989, no pet.) ("Evidence of possible impairment of mental functions is not admissible on the issue of intent [of murder]," *citing Werner* and *Wagner*);

1993 *Cox v. State*, 843 S.W.2d 750, 757 (Tex. App. – El Paso 1992, pet. ref'd) ("denial of a defendant's requested instruction [in a conspiracy to commit capital murder] is not error where the requested instruction ... [pertains to a] ***defensive*** issue which merely denies the existence of an essential element of the State's case") (emphasis supplied); and

1994 *Thomas v. State*, 886 S.W.2d 388, 391 (Tex. App. – Houston [1st Dist.] 1994, pet. ref'd) (in a murder case, "appellant contends that Dr. Quijano's testimony is relevant to the issue whether he intentionally and knowingly killed Larose.... Appellant has attempted to fashion a ***hybrid defense*** to criminal responsibility, one in which he is admittedly sane but unable to form an intent to commit a proscribed act. We do not recognize any such ***defense*** as a legal justification for criminal acts.") (emphasis supplied).

1997 *Warner v. State*, 944 S.W. 2d 812, 815 (Tex. App. – Austin 1997), *pet. dism'd*, 969 S.W.2d 1 (Tex. Crim. App. 1998) (in aggravated kidnaping and assault case, the Austin court held: "We recognize Judge Maloney's concurring opinion in *Penry v. State*, in which he espoused his view that the ***defense of diminished capacity*** is valid in Texas, based on the *Cowles* opinion.... In light of the court of criminal appeals' conclusion in *Wagner*... we are compelled to conclude that ... evidence of mental infirmity [is not admissible] even in cases involving specific intent crimes.") (emphasis supplied).

2001 *Chiles v. State*, 57 S.W.3d 512, 519 (Tex. App. – Waco 2001, no pet.) (The defendant requested a diminished capacity jury instruction be given in the *punishment* phase. Noting that "Texas courts have addressed the issue of diminished capacity only with respect to the guilt-innocence phase of trial," the court also observed that "[t]here is some disagreement regarding the "Cowles exception" but declined to take a position because "[t]his controversy is not relevant to this case.").

2003 *Jackson v. State*, 115 S.W.3d 326 (Tex. App. – Dallas 2003) ("[I]t is clear that ***there is no diminished capacity defense*** to defeat the element of mens rea during the guilt-innocence phase of the trial;... ***there is no "diminished capacity" doctrine in Texas***, it was not deficient representation for appellant's trial counsel to fail to raise diminished capacity or object to its absence."

2003 *O'Dell v. State*, 2003 WL 21047576, *8 (Tex. App. – Eastland 2003, pet. ref'd) (unpublished op.) (Unfortunately for the state of the law, the appellant argued that the evidence was not offered to show diminished capacity... but

In 2005, the Texas Court of Criminal Appeals handed down *Jackson v. State*, 160 S.W.3d 568 (Tex. Crim. App. 2005), and clarified that relevant, mental impairment evidence can be admitted in the guilt/innocence phase of the trial to negate *mens rea*.[21] In this context, the "fact" of brain damage, and concomitant *dis*inhibition of behavioral controls, introduced in the guilt/innocence phase would have a vastly different outcome. By negating the *mens rea* of "intentional" – an essential element for a conviction of capital murder under the TEXAS PENAL CODE §6.03(a)(b), the fact of Mr. Crutsinger's "settled insanity," and concomitant brain damage would exonerate

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that chronic methamphetamine use diminishes the user's mental capacity..." Jackson pointed out at the very beginning of this brief, Diminished Capacity has nothing to do with a defendant's "capacity" to form the requisite intent; it is about whether he, in fact, *did* form it.);

2004 *Atkinson v. State*, 2004 WL 225549 (Tex. App. – Dallas 2004) (unpublished op.) ("Because there is no ***diminished capacity defense*** to defeat the element of mens rea during the guilt-innocence phase of trial, the trial judge did not err in excluding Cook's testimony.").

[21] *See Jackson v. State*, 160 S.W.3d 568, 574-575 (Tex. Crim. App. 2005) ("As with the other elements of the offense, relevant evidence may be presented which the jury may consider to negate the *mens rea* element. And, this evidence may sometimes include evidence of a defendant's history of mental illness."); *Mays v. State*, 223 S.W.3d 651, 654 (Tex. App. – Texarkana 2007, pet. granted) ("*Jackson* makes it clear that, "Texas does not recognize diminished capacity as an affirmative defense i.e., a lesser form of the defense of insanity." *Jackson*, 160 S.W.3d at 573. It does, however, recognize something very closely akin to it, that being based on a diminished-capacity doctrine, this being "simply a failure-of-proof defense in which the defendant claims that the State failed to prove that the defendant had the required state of mind at the time of the offense." Id.")

Mr. Crutsinger altogether under *Evers*, or at a minimum, result in a jury-finding of a lesser included offense, that would never result in a sentence of death.

Given Mr. Crutsinger's settled insanity, he is actually innocent of the offense of capital murder because he lacked the necessary *mens rea.* Accordingly, his conviction of capital murder, and concomitant sentence of death violate fundamental federal constitutional law principles. Habeas relief must be granted. *Herrera v. Collins*, 506 U.S. 390, 417 (1993) ("in a capital case a truly persuasive demonstration of 'actual innocence' made after trial would render the execution of a defendant unconstitutional, and warrant federal habeas relief if there were no state avenue open to process such a claim.").

CONCLUSION

For the reasons set forth above, Mr. Crutsinger is entitled to relief from his unconstitutional conviction and sentence.

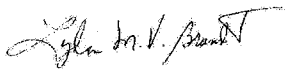
WHEREFORE, Mr. Crutsinger prays that this federal court:

1. Issue a writ of habeas corpus to him brought before it, to the end that he may be discharged from his unconstitutional confinement and restraint and/or relieved of his unconstitutional sentence of death;

2. Grant him one or more hearings, (*see* separately filed motions accompanying this petition), at which he may present any evidence in support of his claims, and allow him a reasonable period of time subsequent to any hearing this court determines to brief the issues of fact and of law raised by this Petition or such hearing.

3. Direct that under Habeas Corpus Rule 7, the record in this case be expanded to include the "additional materials relevant to the determination of the merits of the petition" which are found in exhibits filed with this Petition.

Respectfully submitted,



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REQUEST TO EXPAND THE RECORD

As necessary to demonstrate the validity of each claim in his federal habeas petition, leave is requested to expand the record to include the exhibits attached to his federal habeas petition, as well as any other evidence relevant to this case that was not so included in the state Application for Post-Conviction Writ of Habeas Corpus, including any evidence adduced during discovery and an evidentiary hearing, if the requests for same are granted. *See* Rule 7, Rules Governing Section 2254 Cases in the U.S.D.C.

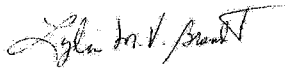
UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF TEXAS
FT. WORTH DIVISION

BILLY JACK CRUTSINGER	)	
Petitioner	)	
	)	**4:07-CV-703-Y**
v.	)	**ECF**
	)	
NATHANIEL QUARTERMAN, Director	)	
Texas Department of Criminal Justice,	)	**(Death Penalty Case)**
Correctional Institutions Division,	)	
Respondent	)	

VERIFICATION

I, Lydia M.V. Brandt, pursuant to 28 U.S.C. § 2242, acting on behalf of Petitioner, certify under penalty of perjury that the foregoing Petition for Writ of Habeas Corpus is true and correct.

Signed this October 29, 2008



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CERTIFICATE OF SERVICE

This certifies that on October 29, 2008, I sent a copy by U.S. mail to

Ellen Stewart-Klein,
Assistant Attorney General
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Paper copies have also been mailed as courtesy copies to:

The Honorable Terry R. Means
U.S. District Court Judge
c/o Clerk's Office
Northern District of Texas, Ft. Worth Division
501 W. 10th Street, Room 310
Ft. Worth, TX 76102-3673

Mr. Billy Jack Crutsinger
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Lydia M.V. Brandt

EXHIBITS

Exhibit A: NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM, *Imagining and Alcoholism: A Window on the Brain*

Exhibit B: HAMDY F. MOSELHY, GEORGE GEORGIOU, AND ASHRAF KAHN, *Frontal Lobe Changes in Alcoholism: A Review of the Literature*, Vol. 36, No. 5, ALCOHOL & ALCOHOLISM 357 (2001)

Exhibit C: Report of Dr. Goodness, dated September 10, 2003

Exhibit D: Letter of Goodness to Ray, dated 06-16-03

Exhibit E: Letter of Dasher to Ray, dated 07-10-03

Exhibit F: Terms of Engagement Contract, dated 7-11-03

Exhibit G: M. C. Brower and B.H. Price, 71 J. NEUROL NEUROSURG. PSYCHIATRY, *Neuropsychiatry of Frontal Lobe Dysfunction in Violent and Criminal Behavior: a Critical Review*, 720, 721 (2001)

Exhibit A

Imaging and Alcoholism: A Window on the Brain

Alcohol Alert From NIAAA

The processes that initiate and maintain alcoholism are regulated by interactions among nerve cells (i.e., neurons) in the brain. These mechanisms interact with emotional, cognitive, and social factors to determine an individual's response to alcohol consumption. The processes that initiate and maintain alcoholism are regulated by interactions among nerve cells (i.e., neurons) in the brain. These mechanisms interact with emotional, cognitive, and social factors to determine an individual's response to alcohol consumption.

Imaging techniques[1] allow scientists to study the link between brain and behavior with minimal risk to the patient. Using imaging, scientists can watch the brain in action as a person performs intellectual tasks, reacts to the environment, or experiences emotions. Data obtained before, during, and after a person has consumed alcohol can be compared and analyzed.

Imaging offers the promise of integrating biomedical, psychosocial, and behavioral aspects of alcoholism, leading to improved prevention and treatment. This Alcohol Alert illustrates some current and potential applications of imaging to alcoholism research.

Alcohol's Effects on Brain Structure and Function

Results of autopsy studies show that patients with a history of chronic alcohol consumption have smaller, lighter, more shrunken brains than nonalcoholic adults of the same age and gender (1).

This finding has been repeatedly confirmed in living alcoholics using structural imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI). Structural imaging reveals a consistent association between heavy drinking and physical brain damage, even in the absence of medical conditions previously considered to be clinical indicators of severe alcoholism (e.g., chronic liver disease or alcohol-induced dementia).

Imaging reveals shrinkage to be more extensive in the folded outer layer (i.e., cortex) of the frontal lobe (2), which is believed to be the seat of higher intellectual functions. In men, vulnerability to frontal lobe shrinkage increases with age (2-4). Current studies will determine if the same effect occurs in women. Repeated imaging of a group of alcoholics who continued drinking over a 5-year period showed progressive brain shrinkage that significantly exceeded normal age-related shrinkage (5). The rate of frontal cortex shrinkage in this study correlated approximately with the amount of alcohol consumed (5).

Shrinkage also occurs in deeper brain regions, including brain structures associated with memory (6-8), as well as in the cerebellum, which helps regulate coordination and balance (9). Limited research suggests that women may be more susceptible than men to alcohol-related brain shrinkage (10,11).

The detection of structural brain damage is complemented by results of functional imaging techniques, such as positron emission tomography (PET) and single photon emission computed tomography (SPECT). By measuring local changes in blood flow and energy metabolism, PET and SPECT can help identify brain regions involved in specific sensory, motor, or cognitive functions. Such studies consistently reveal decreased blood flow and metabolic rates in certain brain regions of heavy drinkers compared with those of nonalcoholics (12,13), even in the absence of measurable shrinkage (14). Structural and functional defects revealed by magnetic resonance spectroscopy (MRS) and PET may reflect a decrease in the number (15) or size (16,17) of neurons or a reduction in the density of communication sites between adjacent neurons (16,17).

Relating Structure and Function to Behavior

A key goal of imaging in alcoholism research is to detect changes in specific brain regions that can be correlated with alcohol-related behaviors. Imaging of the cerebellum has linked both shrinkage (9,18) and decreased blood flow (19) to impaired balance and gait. Such impairment may cause falls among older alcoholics, leading to head injury that may exacerbate brain dysfunction. Studies of cognitive performance, however, have found no consistent relationship between shrinkage of the frontal cortex and impairment of short-term memory and problem-solving (1,20), functions typically disrupted by frontal lobe damage. Conversely, some studies have found an approximate correlation between shrinkage of memory-related brain structures (e.g., mammillary bodies) and the degree of memory impairment (7). Functional imaging studies show that frontal lobe blood flow (21) and metabolism (12) may decrease in alcoholics before significant shrinkage or major cognitive problems become detectable (13,21).

Cognitive functions and motor coordination may improve at least partially within 3 to 4 weeks of abstinence (20) accompanied by at least partial reversal of brain shrinkage (22,23) and some recovery of metabolic functions in the frontal lobes (24) and cerebellum (17,25). Frontal lobe blood flow continues to increase with abstinence, returning to approximately normal levels within 4 years (26). Relapse to drinking leads to resumption of shrinkage (23), continued declines in metabolism and cognitive function (24), and evidence of neuronal cell damage (25).

Mechanisms of Addiction

Studies using animals or cultured slices of brain tissue have identified chemical messengers (i.e., neurotransmitters) and neuronal pathways that may help mediate alcohol's effects. Functional imaging studies are confirming and extending these results. For example, a neuronal pathway involving the neurotransmitter dopamine has been implicated in the development of alcoholism. Nonalcoholic social drinkers administered a mildly intoxicating dose of alcohol (19) and alcoholic subjects experiencing craving for alcohol (27) exhibit decreased blood flow in parts of the brain where dopamine is present. Imaging studies also provide evidence for disrupted response of the neurotransmitter serotonin, which appears to interact with dopamine in the development of alcoholism (28).

Functional imaging reveals that alcoholics have diminished metabolic activity in several frontal brain regions early and late in withdrawal (29,30). In nonalcoholics, benzodiazepine sedatives, some of which are commonly used to treat alcohol withdrawal, produce a temporary alcohol-like impairment of coordination and cognition accompanied by an overall decrease in the brain's metabolic rate. In alcoholics, some frontal brain regions exhibit a smaller metabolic change following benzodiazepine administration than is seen in nonalcoholics. These results may indicate a diminished capacity for dampening excessive neuronal activity, possibly weakening a person's ability to inhibit behavior (30). Among nonalcoholic social drinkers, the effects of benzodiazepines on specific brain regions as assessed by PET (31) and functional MRI (32) differ between

persons with and without a family history of alcoholism. Therefore, an abnormal reaction to benzodiazepines may represent a preexisting risk factor for alcoholism rather than a consequence of long-term alcohol consumption.

A promising application of functional imaging is in the study of cognitive and emotional processes involved in addiction, craving, and relapse. For example, preliminary studies have correlated craving for cocaine with increased metabolism in a neuronal network that integrates emotional and cognitive aspects of memory (33). Similar mechanisms implicated in craving for alcohol may help account for individual differences in vulnerability to alcoholism (34).

Treatment of Alcoholism

Diagnosis. Routine clinical applications of imaging include detecting conditions that commonly co-occur with alcoholism, such as residual brain damage from head trauma (35), various psychiatric disorders (36), and alcohol-induced organic brain disorders characterized by dementia or amnesia (37).

Withdrawal. Up to 15 percent of alcoholics experience seizures during withdrawal, and the likelihood of having such seizures, as well as their severity, increases with the number of past withdrawal episodes. In a structural imaging study of alcoholics who had undergone seizures, Sullivan and colleagues (38) found shrinkage on both sides of the brain behind the frontal lobes. It is not known whether seizures cause the shrinkage or result, in part, from preexisting damage to the area.

Investigators have used PET and SPECT to locate and quantify sites on neuronal surfaces where neurotransmitters implicated in the development of alcoholism interact with the neuron. Results of such research has implicated impaired serotonin function in the severe depression that often accompanies withdrawal (39). Functional imaging is also being used to help evaluate the effects of naloxone on withdrawal-induced craving (40). This medication is chemically related to the anticraving medication naltrexone (ReVia (TM)).

Psychosocial Therapies. Higgins (41) describes the concept of neurobehavioral treatment, which emphasizes learning-based approaches to relapse prevention while paying special attention to the neurobiologic changes that accompany abstinence. For example, a common treatment strategy involves the development of skills for recognizing and coping with environmental influences or emotional states that may induce craving and trigger drinking. This approach requires the ability to monitor and evaluate one's behavior and learn from failed efforts (41). Researchers are using functional imaging to investigate the basis for impairment of these cognitive functions (42).

Appendix: Imaging Techniques

Structural imaging depicts a three-dimensional "slice" of the brain, showing more detail than a conventional X ray. CT is a refinement of x-ray technology, whereas MRI interprets signals emitted by the brain in the presence of a strong magnetic field (43). These techniques are commonly used to help diagnose certain medical conditions (e.g., tumors) as well as in research.

Functional imaging techniques in common use include PET, SPECT, and modifications of magnetic resonance technology (14,43). PET and SPECT provide computer-generated, color-coded, three-dimensional images of the distribution within the brain of radioactive substances injected into the bloodstream. These images can be used to detect changes in blood flow in specific brain regions or to determine the locations of various neurotransmitters or receptors. Functional MRI can locate and assess levels of brain activation associated with

motor, sensory, or cognitive processes that may be impaired by alcohol over time at intervals as short as a few seconds (44). MRS can detect specific molecules, including alcohol itself (45), and can detect metabolic changes underlying deterioration of neuronal structural integrity (43).

Imaging and Alcoholism: A Window on the Brain—A Commentary by NIAAA Director Enoch Gordis, M.D.

Imaging technology has helped alcohol researchers to study how alcohol damages internal organs, such as the brain and the liver. More recent advances in imaging techniques are allowing investigators to also study alcohol dependence itself. Scientists are beginning to measure alcohol's effects on mood, emotional states, craving, and cognition while simultaneously assessing metabolic, physiologic, and neurochemical function in the brain. These innovations in imaging technology will help not only the alcohol field, but also all fields of medicine where biology and behavior are so closely linked.

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[1]See appendix for brief descriptions of specific imaging techniques.

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